# Comparison of Lung Sounds and Gas Trapping in the Study of Airway Mechanics

by David G. Frazer,\* Louis D. Smith,\* Leo R. Brancazio,\* and Kenneth C. Weber\*

We have previously shown that gas is trapped in isolated animal lungs and have proposed that this gastrapping process is related to meniscus formation in the small airways of the lung. The purpose of this investigation was to compare how the lung sound-generation process and the gas-trapping process are related to airway mechanics and each other. Rats were anesthetized, the heart and lungs were removed en bloc and placed in a liquid-filled plethysmograph. Lung sounds were recorded by using a microphone acoustically coupled to the tracheal cannula. The results show that discontinuous lung sounds in the form of crackles occur during lung inflation at the same time gas trapping takes place.

# Introduction

For the past several years we have been investigating the process responsible for gas trapping in isolated lungs. The amount of gas trapped in the lung during an inflation-deflation cycle increases as the inflation rate decreases (1,2), as lung volume increases (2), as the minimum transpulmonary pressure decreases (3), as the diffusivity of the gas used to ventilate the lung increases (4), and as the wet weight/dry weight of the lung increases (5).

In addition, we have shown that gas trapping occurs in most animal species, including rats, mice, rabbits, hamsters, and cats (6) and in dogs, sheep, pigs, and guinea pigs (unpublished observations). In one animal species, the rat, we have shown that the gas-trapping mechanism in live anesthetized animals has the same characteristics as the gas-trapping processes in isolated lungs (7).

One might expect that the breaking and possibly the formation and movement of menisci in small airways could generate lung sounds. If this is true, the mechanical events which are associated with the gas trapping process may be recognized by specific acoustical characteristics. The advantage of using an acoustical method rather than directly measuring the amount of gas trapped in the lung is that acoustical methods are generally noninvasive while trapped gas measurements are not. In a disease such as byssinosis, for example, where the amount of fluid in the airway has been shown to change (8,9), the gas-trapping (meniscus formation) process would also be expected to change significantly (5). If the production of lung sounds parallels the alterations in gas

trapping, it would be possible to monitor the fluid content of the airways as live animals and humans are exposed to cotton dust.

The goal of this study was to correlate lung sounds with past gas-trapping experiments in isolated rat lungs. Once this correlation is made, it should be possible to measure lung sounds in animals and humans exposed to cotton dust and obtain a valid prediction of peripheral airway function changes.

# **Methods**

Long-Evans hooded male rats weighing between 250 and 350 g were anesthetized with sodium pentabarbitol (65 mg/kg) and exsanguinated via the abdominal aorta. The heart and lungs were removed en bloc, then degassed according to the method of Stengel et al. (10) and placed within a liquid-filled plethysmograph as shown in Figure 1. Lungs were inflated-deflated by raising and lowering the pressure reference chamber. The level of the fluid in the pressure reference chamber with respect to the lung carina was equal to transpulmonary pressure. Changes in lung volume  $(V_L)$  were determined by measuring the amount of air displaced by the saline in the reference chamber with a minispirometer (Med. Sciences Model #118). Transpulmonary pressure  $(P_L)$  was measured with a differential pressure transducer (Setra model #233E). Lung sounds were detected with a microphone (B&K model #4166) acoustically coupled to the trachea. Lung volume and lung sounds were recorded versus transpulmonary pressure on a dual channel oscilloscope (Tektronics model #564). Lung sounds were simultaneously measured with a fast Fourier spectrum (FFT) analyzer (B&K model #2033) and a precision integrating sound level meter (B&K model #2218). The FFT analyzer was

<sup>\*</sup>Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health, Morgantown, WV 26505.

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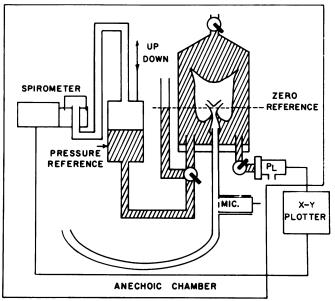


FIGURE 1. Experimental setup for measuring lung sounds as an excised lung was inflated-deflated in a liquid-filled plethysmograph by lowering-raising a reference chamber.

used to determine either the spectrum of individual crackles or the average spectrum of a large number of crackles while the precision integrating sound level meter was used to determine the accumulative sound energy level measured at the trachea as the lung was inflated-deflated.

## **Previous Gas-Trapping Experiments**

A cycle was defined as a single inflation—deflation of the lung. Six pressure-volume,  $P_{\rm L}-V_{\rm L}$ , curves were recorded for both the normal and edematous lungs. Each curve consisted of four cycles at an inflation—deflation rate of 3.82 cm³/min. During the first cycle, lungs were inflated from 0.0 cm  $\rm H_2O$  to 30 cm  $\rm H_2O$  [ $P_{\rm L}(\rm max)$ ] then deflated to preselected end-expiratory pressures [ $P_{\rm L}(\rm min)$ ]. In cycles 2 and 3, lungs were inflated-deflated between  $P_{\rm L}(\rm min)$  and  $P_{\rm L}(\rm max)$ . In the fourth cycle the lungs were again inflated from  $P_{\rm L}(\rm min)$  to  $P_{\rm L}(\rm max)$  but were then deflated to -5 cm  $\rm H_2O$ .  $P_{\rm L}(\rm min)$  was +6 cm  $\rm H_2O$  for the first  $P_{\rm L}-V_{\rm L}$  curve then reduced to +4, +3, +2, 0.0, and -5 cm  $\rm H_2O$  for the second through sixth  $P_{\rm L}-V_{\rm L}$  curves.

A variable, proportional to the fraction of gas trapped in the lungs following a four-cycle  $P_{\rm L}-V_{\rm L}$  curve, was calculated by dividing the amount of gas trapped in the lungs at -5 cm  $\rm H_2O$  ( $V_{\rm m}$ ) following the fourth inflation-deflation cycle ( $V_{\rm max}$ ). The variable ( $V_{\rm m}/V_{\rm max}$ ) was equal to the normalized minimum volume of the lungs. By recording a series of four-cycle  $P_{\rm L}-V_{\rm L}$  curves for each lung in which  $P_{\rm L}$ (min) was held at different values, it was possible to determine the relationship between  $P_{\rm L}$ (min) and  $V_{\rm m}/V_{\rm max}$  for that lung. This method has been previously discussed in detail (3).

# **Lung Sound Experiments**

In this study a 12-cycle  $P_{\rm L}-V_{\rm L}$  curve was recorded for each lung. During the initial cycle a lung was inflated to 30 cm  $\rm H_2O$  [ $P_{\rm L}(\rm max)$ ] then deflated to an end-expiratory pressure [ $P_{\rm L}(\rm min)$ ] of +6 cm  $\rm H_2O$ . In cycles 2 to 12, the lung was inflated to  $P_{\rm L}$  max and deflated in order to values of  $P_{\rm L}(\rm min)$  of +5, +4, +3, +2, +1, 0, -1, -2, -3, -4, and -5 cm  $\rm H_2O$ . During each of the twelve cycles the total accumulative sound power was measured with a microphone acoustically coupled to the trachea. The total linear sound power  $S_{\rm T}$ , was calculated by using equation (1); the total sound energy  $L_{\rm ax}$ , in decibels, was calculated using equation (2)

$$S_{\rm T} = \int_0^T [P(t)/P_0]^2 dt \tag{1}$$

$$L_{\text{ax}} = 10 \log \int_0^T [P(t)/P_0]^2 dt$$
 (2)

where P(t) is the instantaneous sound pressure measured at the microphone,  $P_0$  is the sound pressure reference, and T is the total time to inflate the lung starting at zero time

In a separate experiment one lung was inflated-deflated for 10 cycles between a  $P_{\rm L}({\rm max})$  of  $-30~{\rm cm~H_2O}$  and a  $P_{\rm L}({\rm min})$  of  $-5~{\rm cm~H_2O}$ . Both lung volume and instantaneous sound pressure level were recorded versus transpulmonary pressure on a dual trace oscilloscope as trapped gas continued to accumulate in the lung. Individual discontinuous lung sounds (crackles) contained within the instantaneous sound pressure level envelope were also examined and their sound power spectra analyzed.

# **Results**

# **Previous Gas-Trapping Experiments**

The normalized amount of gas trapped in the lung  $(V_{\rm max})$  plotted versus end-expiratory pressure is shown in the upper panel of Figure 2. The continuous curve drawn through the points represents the best least-square fit of the integral of the normal curve from  $+\infty$  to  $P_{\rm L}({\rm min})$  as  $P_{\rm L}({\rm min})$  approaches  $-\infty$ . It has been proposed that the change in  $V_{\rm max}/V_{\rm max}$  with respect to  $P_{\rm L}({\rm min})$  reflects the relative number of airways in which menisci have formed as the lungs were deflated from  $P_{\rm L}({\rm max})$  to  $P_{\rm L}({\rm min})$  (3,5).

# **Lung-Sound Experiments**

When the linear accumulative sound power was plotted versus end-expiratory pressure for the 12-cycle  $P_{\rm L}$ – $V_{\rm L}$  curves, the results in the lower panel of Figure 2 were obtained. The right ordinate shows sound power plotted on a linear scale  $S_{\rm T}$  and the left ordinate shows sound power level on a decibel scale,  $L_{\rm ax}$ .

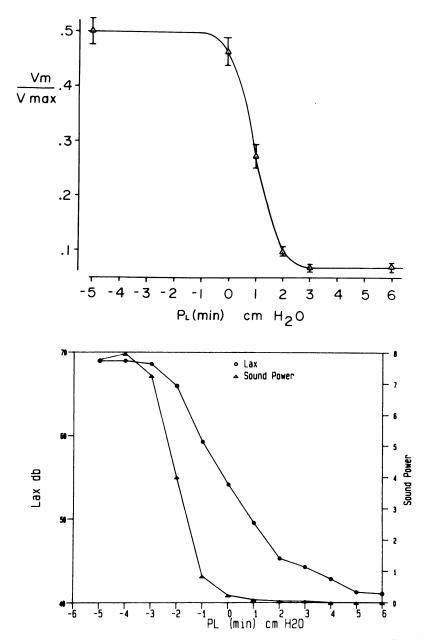


FIGURE 2. The amount of trapped gas as a function of end-expiratory pressure is shown in the top panel. In the lower panel the total sound energy is expressed on both a linear scale and a dB scale  $(L_{ax})$ .

In order to determine when sound energy was generated in the lung during lung inflation, sound pressure level was recorded versus  $P_{\rm L}$  during lung inflation for 10 continuous inflation—deflation cycles. Figure 3 shows the results for the second cycle (upper panel) and the tenth cycle (lower panel). The envelope of the sound pressure level varied as the lung was inflated and changed shape as the lung accumulated trapped gas. Lung sound energy was a maximum near the knee of the inflation curve but continued until maximum lung volume was reached. These results indicate that lung units most likely continue to be recruited until lung volume reaches total lung capacity at  $P_{\rm L}({\rm max})$ . There was no detectable sound energy generated in the lung during deflation on any cycle until

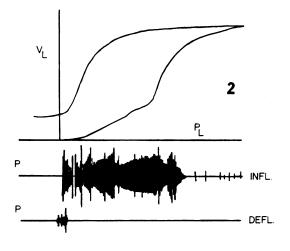
low transpulmonary pressures were reached.

When the sound signal was examined on an expanded time scale, it could be seen that the signal consists of a series of discrete discontinuous sounds or crackles. A single crackle plotted versus time is shown in the upper panel of Figure 4. The lower panel shows the spectral density of the sound energy contained in the crackle expressed in decibels. It should be noted that the crackle contains significant frequency components up to 8 kHz.

# **Discussion**

In the past we have shown that the rate of inflation greatly affects the amount of trapped gas in a lung, as

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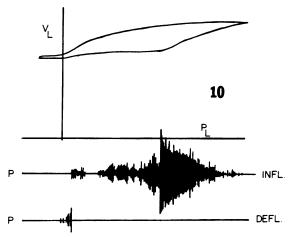


FIGURE 3. Results of a representative experiment in which the lung was ventilated between -5 cm  $\rm H_2O$  and + 30 cm  $\rm H_2O$  for 10 continuous cycles. The second  $P_{\rm L}{-}V_{\rm L}$  cycle is shown in the upper panel and the tenth  $P_{\rm L}{-}V_{\rm L}$  cycle is shown in the lower panel. Note that the lung sounds were not present on deflation.

it is inflated-deflated while the deflation rate plays only a minor role (2). When the inflation-deflation rate was reduced, the amount of gas trapped in the lung increased. We have also presented evidence in the past that gas diffuses through a liquid barrier such as a miniscus to enter the trapped gas space (1,4).

One of our most important observations, however, was that end-expiratory pressure,  $P_{\rm L}({\rm min})$ , has a large effect on the volume of gas trapped in the lungs (2,3,5). Our explanation of this finding is that gas is trapped only during lung inflation if liquid films or menisci form across the airways during lung deflation. Ventilating the lungs with a positive end-expiratory pressure keeps the dimensions of the airways sufficiently large that the formation of menisci in the lumen of the airways is inhibited. By recording a series of pressure–volume curves with different end-expiratory pressures, it is possible to obtain the relationship between trapped gas volume (or

meniscus formation) and end-expiratory pressure (Fig. 2A, upper panel).

Surprisingly, the results of this study indicated that menisci formed in the lung during deflation between end-expiratory pressures of +4 and 0.0 cm  $\rm H_2O$  since the experiments of Cavagna et al. (11) and Hughes et al. (12) showed that airways do not physically buckle until negative transpulmonary pressures of approximately -3 cm  $\rm H_2O$  are reached. It is likely, therefore, that airway closure is related to two separately occurring events as lungs are deflated. These events are the formation of a meniscus across the airway at positive transpulmonary pressures followed by a mechanical buckling of the airway wall at negative values of  $P_{\rm L}$ .

The object of this study was to determine how lung sounds which are generated during lung inflation in excised lungs are related to the airway closure and reopening process.

The acoustical energy measured at the trachea during lung inflation following lung deflation to different endexpiratory pressures is presented in Figure 2. These results show that the total sound energy level resulting from the summation of all the discontinuous lung sounds. or crackles, increased as the end-expiratory pressure decreased. Very little acoustical energy was detected at the trachea during lung deflation. The range of end-expiratory pressures over which  $L_{ax}$ , total sound energy level expressed in decibels, increased as end-expiratory pressure decreased was very similar to that found for the gas trapping experiments. The range of end-expiratory pressures over which linear sound power changed was much lower than for  $L_{\rm ax}$  (Fig. 2). These results raise the question of which method of expressing sound energy most accurately represents the gas-trapping events which are simultaneously occurring in the lung.

We believe that the explosive sounds, or crackles, which are recorded during lung inflation result from the sequential opening of lung units as described by Frazer and Franz (13). In the past, crackles have been classified as being either coarse or fine. Coarse crackles are considered to have a greater amplitude and lower frequency content than fine crackles (14) and are believed to be generated in the larger more central airways. Fine crackles are thought to be generated in the lung periphery.

When lung sounds were measured at the trachea in this study the magnitude of the individual crackles diminished as the lung was inflated (Fig. 3, upper and lower panels). Two reasons for this might be: (1) coarse crackles occur initially at lower transpulmonary pressure as the larger lung units open first, whereas fine crackles occur at higher transpulmonary pressures when the smaller peripheral lung units open, and (2) crackles generated by events occurring in the large airways nearer the microphone appear to have a greater amplitude than those occurring in the periphery of the lung.

There is evidence (15,16) that the gas-trapping process occurs predominantly in the periphery of the lung, so that it is likely that fine crackles rather than coarse crac-

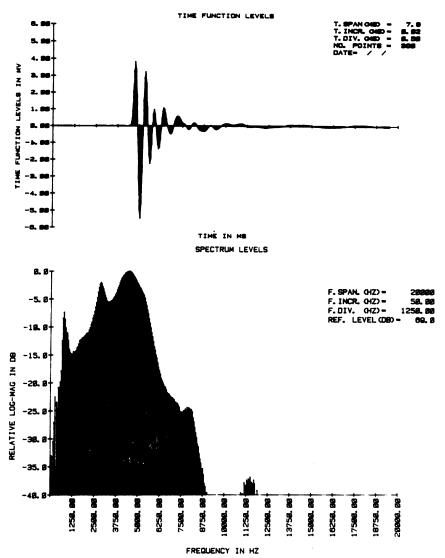


FIGURE 4. The upper panel shows sound pressure for a single crackle measured at the trachea as a function of time. The lower panel shows the power spectrum of the same crackle. Note that there was significant energy at frequencies greater than 2 kHz.

kles occur in the same region of the lung where gas trapping takes place. It should be noted at this point that the total linear sound energy measurement is influenced much more by the larger acoustical events represented by the coarse crackles in the major airways than by the fine crackles in the periphery of the lung. The logarithm of sound power  $L_{\rm ax}$  tends to weigh the contribution of coarse and fine crackles more evenly. Thus, the linear sound power measurement would be expected to be representative of mechanical events occurring in the large airways while  $L_{ax}$  would emphasize information related to the gas trapping process in the periphery of the lung. Since  $L_{ax}$  and gas trapping appear to be similarly related to end-expiratory transpulmonary pressure, it could be concluded that trapped gas measurements and  $L_{\rm ax}$  measurements may give information concerning the same processes occurring in the periphery of the lung.

Previous investigators have suggested that the source

of fine crackles is the explosive reopening of atelectatic regions of the lung (17). Results of this study show, however, that the tracheal sounds did not diminish as trapped gas accumulated in the lung and reached a volume of 75% TLC (compare the upper and lower panels of Fig. 3). It appears likely, therefore, that crackles are related to the sequential reopening of lung units which may either be atelectatic or contain trapped gas.

In summary, we have shown in the past that examination of the gas-trapping process can be used to obtain a very sensitive index for determining small alterations in the periphery of the lung brought about by disease (5,18). The usefulness of these particular methods were limited, however, because they are only applicable for excised lungs. If the same information can now be obtained noninvasively by tracheal lung sound analysis, it will be possible to use this new technique for both live animals and man. Studying the tracheal lung sounds of

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laboratory animals exposed to cotton dust will be especially helpful in determining the progression of small airway obstruction during the onset of byssinosis.

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